

# FACTSHEET

## *Veterinary Services*

United States  
Department of  
Agriculture

Animal and  
Plant Health  
Inspection  
Service

September 1999

## Transmissible Mink Encephalopathy

Transmissible mink encephalopathy (TME) is a rare illness that affects the central nervous system of ranch-raised mink. It was first detected in the United States in 1947. Since then, TME outbreaks have been reported in numerous locations worldwide, including the United States, Canada, Finland, Germany, and the republics of the former Soviet Union.

### Related Diseases

TME is classified as a transmissible spongiform encephalopathy (TSE). Different TSE's are caused by similar as-yet uncharacterized agents that produce spongiform changes in the brain. Other TSE's include scrapie, which affects sheep and goats; bovine spongiform encephalopathy (BSE); feline spongiform encephalopathy; chronic wasting disease of deer and elk; and kuru, both classical and variant Creutzfeldt-Jakob disease (CJD), Gerstmann-Straussler-Scheinker syndrome, and fatal familial insomnia, five rare diseases in humans. TSE's have also been reported in Europe in captive wild ruminants, cats, and monkeys. The occurrence of TSE's in captive wild animals is believed to have resulted from BSE-contaminated feed.

These rare, progressively degenerative central nervous system diseases are characterized by a very long incubation period, a short clinical course, and a 100-percent mortality rate. The infectious agent responsible for TME is smaller than the smallest known virus and has not been characterized to date. There are three main theories on the nature of this agent: (1) the agent is a virus with unusual characteristics, (2) the agent is a prion—an exclusively host-coded protein that is modified to a protease-resistant form after infection, or (3) the agent is a virino—a small, noncoding regulatory nucleic acid coated with a host-derived protective protein. The TME agent is extremely resistant to heat and to normal sterilization processes. It also does

not evoke any detectable immune response or inflammatory reaction in host animals.

Public interest surrounding TSE's soared when the United Kingdom announced in March 1996 that BSE may be linked to a variant form of CJD. BSE, widely referred to as "mad cow disease," has devastated the cattle industry in the United Kingdom. The fear of this disease prompted countries around the world to step up measures to ensure that they remain free of BSE. BSE has not been detected in the United States, and the U.S. Department of Agriculture (USDA) works proactively to keep it that way. USDA's Animal and Plant Health Inspection Service (APHIS) and Food Safety and Inspection Service take aggressive measures in prevention, education, surveillance, and response.

### Clinical Signs

TME has an average incubation period of more than 7 months before the onset of clinical signs. These signs can last from 3 days to 6 weeks. Early clinical signs, which can be quite subtle, include an increase in nest soiling and dispersal of droppings throughout the cage. In addition, mink may step into their food often or eat with difficulty. As the disease progresses, an infected animal becomes increasingly excited, arching its tail over its back like a squirrel. TME-infected animals may exhibit severe incoordination, difficulty walking, and pronounced jerkiness of hind limbs. In advanced cases, signs include rapid circling, compulsive chewing of the tail, and clenching of the jaw. Seizures rarely occur. Near death, affected mink become sleepy and unresponsive.

### Diagnosis

TME produces no changes in the body that are visible upon necropsy examination. However, microscopic examination shows that the disease is limited to the central nervous system, causing distinct spongelike changes in specific areas of the brain.

Currently, there are no validated tests to detect TSE's in a live animal. Veterinary pathologists confirm disease by microscopic examination of brain tissue or by the detection of the prion protein.

## Epidemiology

Epidemiologic studies suggest that animals contract the disease by external exposure to the infectious agent, such as by eating contaminated feed. No evidence suggests that the TME agent spreads by contact between unrelated mink or from mother to nursing young. The disease has been identified in both genders and all color phases in animals greater than 1 year old.

The first documented TME outbreak in the United States occurred in 1947 on one ranch in Wisconsin and then on a ranch in Minnesota that had received mink from the Wisconsin ranch. In 1961, TME outbreaks occurred on five ranches in Wisconsin. In 1963, outbreaks occurred in Idaho, Minnesota, and Wisconsin. Epidemiologic data from the Minnesota and Wisconsin outbreaks trace the cases in those States to one common purchased food source.

## The 1985 Stetsonville Outbreak

The most recent TME outbreak occurred on one mink ranch in Stetsonville, WI, in 1985. In the herd of 7,300 adult mink, 60 percent of the animals died. Clinical signs included tail arching, incoordination, and hyperexcitability. At the most advanced stages of the disease, the animals were in trancelike states and eventually died.

The outbreak lasted 5 months. Microscopic examination of sections of the brain confirmed the spongeliike changes characteristic of TME. Diagnostic tests identified the prion protein. The following year, mink born during the outbreak showed no signs of TME.

The late Richard Marsh, a veterinary virologist at the University of Wisconsin who studied the transmission of TME and other TSE's, investigated this outbreak. Marsh learned that the mink were fed a diet composed of fresh meat products from "downer cattle" and commercial sources of fish, poultry, and cereal. Downer cattle are nonambulatory and cannot rise because they are affected with a condition such as a metabolic disease, broken limbs, or central nervous system disorder. Marsh theorized that the meat from these downer cattle introduced a TSE agent to the mink in which TME resulted.

Although Marsh's hypothesis is based on speculation and anecdotal evidence, in 1993 APHIS adjusted its national BSE surveillance program to include testing downer cattle for evidence of a TSE. The brains of more than 8,400 cattle have been examined at APHIS' National Veterinary Services Laboratories and other State diagnostic laboratories. Not a single tissue sample has revealed evidence of BSE or another TSE in cattle.

## Additional Information

Veterinarians and livestock and poultry owners who suspect an animal may be affected with a TSE should immediately contact State or Federal animal health authorities.

For further information, contact

USDA, APHIS, Veterinary Services  
Emergency Programs  
4700 River Road, Unit 41  
Riverdale, MD 20737-1231  
Telephone: (301) 734-8073  
Fax: (301) 734-7817

Current information on animal diseases and suspected outbreaks is also available on the Internet. Point your Web browser to <http://www.aphis.usda.gov> to reach the APHIS home page.

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